*Case Reports*

# Mitral Valve Regurgitation Causing Right Upper Lobe Pulmonary Edema

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*When radiography is performed in patients with mitral regurgitation, cardiogenic pulmonary edema is a typical finding; however, asymmetric pulmonary edema has also been reported. We describe the case of a patient in whom mitral valve regurgitation caused isolated pulmonary edema in the right upper lung. We include a discussion of pulmonary edema in conjunction with mitral regurgitation.* ***(Tex Heart Inst J 2001;28: 53-6)***

Local alterations of vascular homeostasis are a cause of asymmetric pulmonary edema. This often occurs in the presence of an infectious infiltrate. However, there are many other causes of asymmetric edema, as described by Calenoff and colleagues.[1] Their review reinforces the importance of evaluating all clinical aspects of the patient's condition and knowing the pathophysiology of edema to arrive at a correct diagnosis.

Mitral valve regurgitation is understood to cause pulmonary edema as a result of increased intravascular hydrostatic pressure. Previous studies have shown that predominantly right upper lobe edema may be caused by a directed regurgitant jet with resultant local increases in hydrostatic pressure.[2] The case of our patient presents a good example of these findings.

## Case Report

The patient was a 58-year-old nonsmoking man who presented at the emergency room with shortness of breath in December of 1999. He reported a medical history of recurrent bronchitis (in the distant past) that had been treated effectively with antibiotics by his primary care physician. He had also been diagnosed in 1984 with mitral valve prolapse, which was associated with occasional acute episodes of mitral regurgitation. The patient had been in his usual state of health until 10 to 14 days prior to admission to the hospital. One week before admission, he developed dyspnea that increased considerably during the 2 days before he came to the emergency room. He had a cough with some sputum production, which contained occasional flecks of blood. The man was employed as a grade school teacher, and he stated that several of his students had been ill. However, he had not experienced any noticeable fevers, chills, or sweats, and he was having chest pain only when he coughed.

In the emergency room, the patient appeared to be in mild respiratory distress. His temperature was 99.3 °F; his respiratory rate was 20 breaths/min on oxygen by nasal cannula; blood pressure, 110/60 mmHg; and pulse, 112 beats/min in sinus tachycardia. On auscultation, fine crackles were heard over the right anterior chest, and there was a grade 3/4 holosystolic murmur radiating to the apex of the heart.

Laboratory tests revealed a hemoglobin of 13.4 g/dL (normal range, 14 to 18 g/dL) and a white blood cell count of 11,230/mm$^3$ (normal range, 4,000 to 10,800/mm$^3$) without elevated band neutrophils. The albumin level was 3.8 g/dL (normal range, 3.2 to 5.5 g/dL); lactate dehydrogenase, 716 U/L (normal range, 110 to 200 U/L); erythrocyte sedimentation rate, 47 mm/h (normal range, 0 to 10 mm/h); and troponin I, 0.2 ng/mL (normal range, 0 to 1.5 ng/mL), with follow-up values of 0.2 and <0.2 ng/mL, respectively. Blood, urine, and sputum cultures were obtained. Chest radiographs performed elsewhere revealed extensive

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opacification of the right upper lobe with bilateral pulmonary vascular congestion. There was no evidence of pleural fluid (Fig. 1).

After a pulmonary consultation, the patient began treatment for possible atypical pneumonia, and evaluation was continued. The patient's initial troponin level was negative, but because of the time course and known medical history, infarction-induced exacerbation of mitral regurgitation remained a possibility. An echocardiogram performed on the day of admission indicated an ejection fraction of 0.65, mild left ventricular enlargement, moderate left atrial enlargement, and a probable ruptured chorda tendineae resulting in severe mitral regurgitation. Follow-up transesophageal echocardiography confirmed the presence of a ruptured chorda that was causing a posterior valve leaflet to be partially flail.

The patient underwent mitral valve repair. He recovered postoperatively with the administration of diuretics, and his right upper lobe edema resolved (Fig. 2). All cultures obtained during the period of hospital admission were negative for infectious organisms. Six months after surgery, the patient remained asymptomatic.

## Discussion

Pulmonary edema is accumulated fluid within the extravascular compartments of the lung. The shift of fluid from the intravascular to the extravascular compartment is most frequently the result of increased pulmonary venous pressure or increased capillary permeability. Less often, decreased oncotic pressure within the plasma and lymphatic insufficiency cause pulmonary edema.[3] Initial alterations in any of these factors can cause interstitial edema; more pronounced changes lead to alveolar edema.

When pulmonary edema is diagnosed, radiologists try to distinguish between cardiogenic (hydrostatic) edema and noncardiogenic edema, because of the usefulness of such a distinction in patients whose conditions are difficult to evaluate clinically. This differentiation can be problematic, but there are certain radiographic findings which, in conjunction with the clinical history, may support a diagnosis.[3] The distribution of pulmonary edema and blood flow, and the width of the vascular pedicle are a few of the radiographic features that should be noted.

Cardiogenic pulmonary edema, like noncardiogenic, can be both interstitial and alveolar, depending on the degree of alteration of the intravascular homeostasis. Initially, chest radiographs of cardiogenic edema show peribronchial cuffing, Kerley lines, and subpleural fluid that is most pronounced centrally.[4] In addition, several other findings may be present, including enlargement of the vascular pedicle (upper mediastinal width), redistribution of blood flow in an upper lobe, and pleural effusions.[3] Air bronchograms are considered a rare finding in cardiogenic edema.[3] Using several such variables, 1 group[4] achieved an accuracy of 81% to 91% in determining the cause of pulmonary edema. This type of diagnostic study requires great attention to detail and may not be critical in patients whose conditions are relatively easy to evaluate clinically.

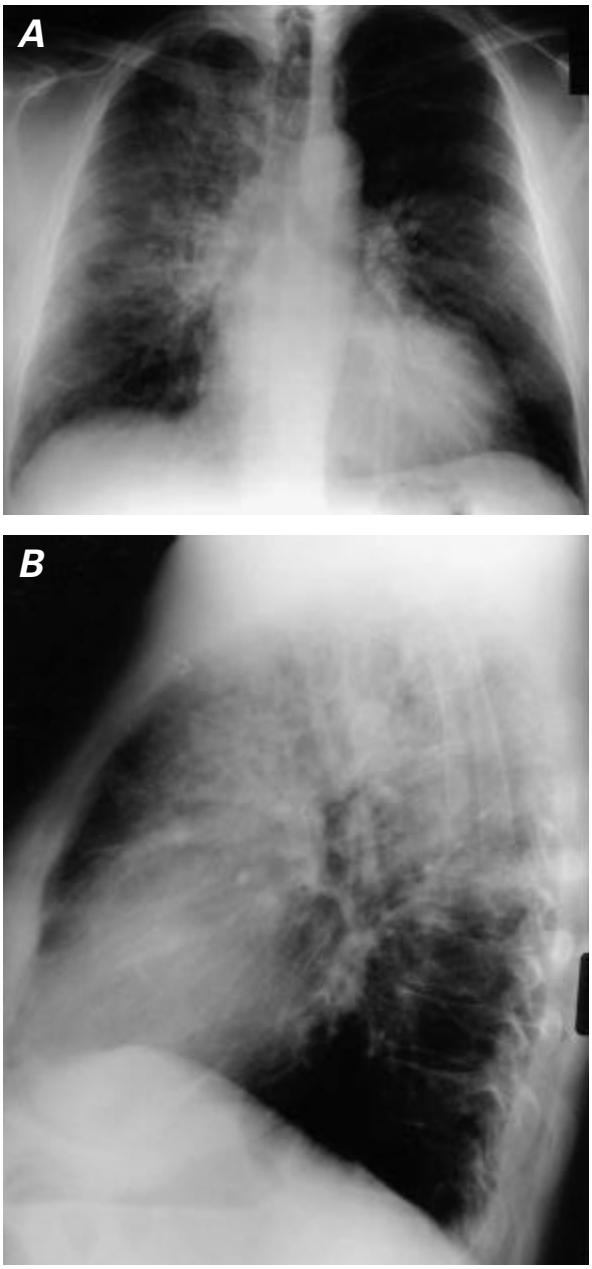


***Fig. 1*** *Posteroanterior (**A**) and lateral (**B**) chest radiographs obtained before the patient presented at the emergency room, revealing right upper lobe pulmonary edema. Left atrial enlargement is suggested and the pulmonary vasculature appears prominent bilaterally. There are no pleural effusions.*

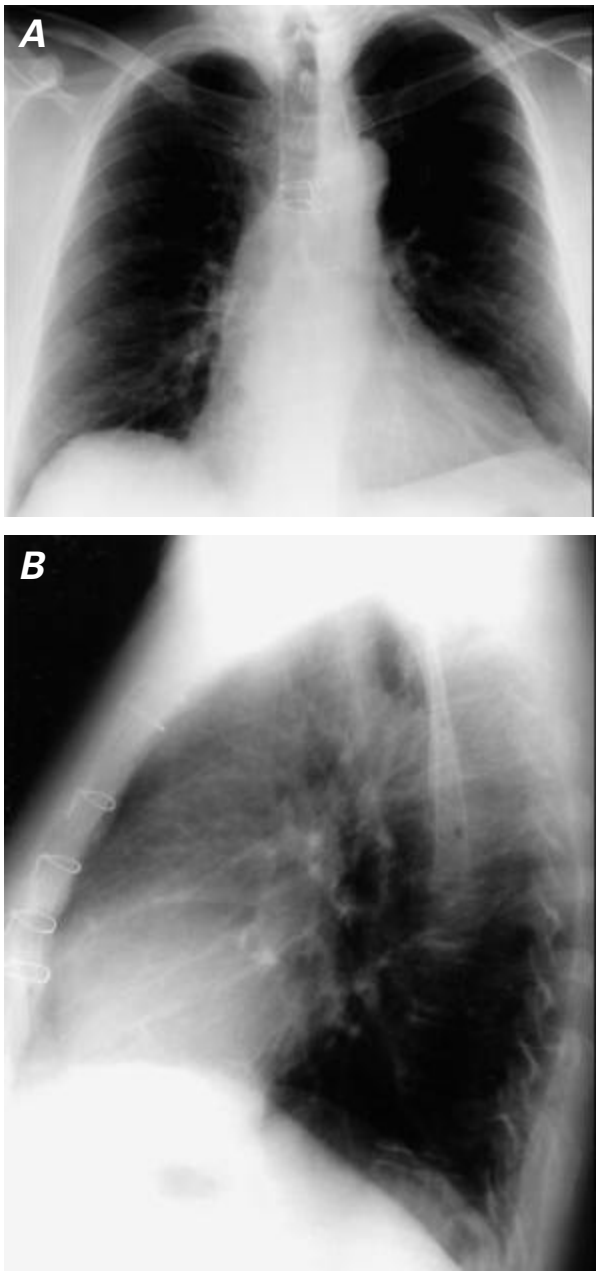


***Fig. 2*** *Follow-up posteroanterior (**A**) and lateral (**B**) chest radiographs, revealing resolution of the pulmonary edema and vascular congestion. The patient has undergone a mitral valve repair; the median sternotomy wires are evident.*

In a recent radiographic review, Glueker and coauthors[5] divided pulmonary edema into 4 pathophysiologic types based on altered hydrostatic pressure, altered permeability, and alveolar damage. Categorizing the specific findings into these types from a chest radiograph may not always enable a diagnosis, but it can help to limit the differential. The authors emphasized the need for clinical correlation to aid in diagnosis.

Asymmetric pulmonary edema is found in patients with various clinical conditions. A review noting the distinction between ipsilateral edema and unilateral edema occurring on the contralateral side was published in 1978.[1] Those authors noted that ipsilateral pulmonary edema is edema that occurs on the same side as a focal insult to 1 lung, without evidence of edema in the other lung. Causes of this type of edema include bronchial obstruction, veno-occlusive disease, unilateral aspiration, pulmonary contusions, and several others. Although it was not mentioned specifically, unilateral edema caused by mitral regurgitation would be included in this category. The 2nd classification, contralateral pulmonary edema, was described by the authors as edema that occurs in a normal lung only because of an abnormality of the opposite lung. This occurs with congenital absence of a pulmonary artery, Swyer-James syndrome, pulmonary embolism, and lobectomy, among other conditions. In general, edematous fluid collects only in the contralateral lung, because there is either no blood flow or no lung parenchyma ipsilaterally. Other authors[5] have suggested that asymmetric edema is also seen with the fibrotic changes that occur in conjunction with tuberculosis, sarcoidosis, and asbestosis. The position of the patient can also influence the distribution of pulmonary fluid.

The pulmonary edema associated with mitral valve regurgitation is usually cardiogenic;[6] occasionally, however, it is isolated or predominantly right upper lobe pulmonary edema.[7-9] Schnyder and coworkers[10] reported a 9% prevalence of predominantly right upper lobe pulmonary edema in cases of severe mitral valve regurgitation. This percentage emphasizes the need for the differential diagnosis of isolated right upper lung edema to include mitral valve disease. The initial diagnosis of pneumonia is often made incorrectly, because the possibility of cardiac disease is not considered.[10]

Right upper lobe pulmonary edema may seem to remain outside the aforementioned pathophysiologic principles. However, ultrasound has been used to define the mechanism of isolated edema on the basis of these principles.[2,11] In 1982, Miyatake and associates[11] showed that the direction of the regurgitant jet caused by flail mitral valve leaflets varies depending on the leaflet involved. Roach's group[2] later reported that the jet of regurgitant flow in a patient with a flail posterior valve leaflet is directed specifically toward the right pulmonary vein. Further evidence of a pressure differential between the right and left pulmonary veins confirms the finding. Therefore, right upper lobe edema is most likely the result of an asymmetric increase in the hydrostatic pressure of the right upper lung caused by asymmetric blood flow from the left atrium.

The case presented herein is a very good example of the clinical, pathophysiologic, and radiographic features of right upper lobe pulmonary edema caused

by mitral valve regurgitation. Our patient initially presented with a confusing medical history, equivocally suggesting pneumonia. Moreover, radiography revealed focal right upper lobe edema that was at first thought to be consistent with pneumonia. However, the pattern of cardiogenic edema with central vascular prominence was also present. Echocardiography confirmed a flail posterior leaflet with severe mitral valve regurgitation, and the patient responded well to mitral valve repair. Our case emphasizes the need to include mitral valve regurgitation in the differential diagnosis of right upper lobe pulmonary edema.

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